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LETTER TO EDITOR

What Emergency Medicine Specialists Need to Know about Hantavirus: A Letter to the Editor

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On May 2, 2026, a deadly outbreak was reported on a cruise ship in the Atlantic Ocean. The World Health Organization (WHO) confirmed that hantavirus was responsible for the outbreak, which resulted in hantavirus pulmonary syndrome (HPS) (1).

Hantaviruses are a family of viruses transmitted primarily by rodents. Human infection occurs through exposure to infected rodent urine, saliva, or droppings, most commonly from rats and mice (2).

The risk of infection is highest among individuals who clean, work, play, or live in environments contaminated with dried rodent urine or feces. In rare cases, hantavirus infection may also occur through rodent bites or scratches or through ingestion of contaminated food. Person-to-person transmission is uncommon and has been reported only with the Andes virus strain (3).

Symptoms typically begin approximately 1 week after viral exposure but may take up to 8 weeks to appear (4).

In the Western Hemisphere, hantaviruses can cause HPS. Patients typically present initially with nonspecific flu-like symptoms, including fever, chills, fatigue, myalgia, and headache. Many patients also develop gastrointestinal symptoms such as abdominal pain, nausea, vomiting, or diarrhea (2,4). Approximately 4–10 days after symptom onset, patients may develop cough and dyspnea that can rapidly progress to severe respiratory failure and critical illness (4). Patients with fatal HPS often demonstrate severe myocardial depression that may progress to sinus bradycardia, electromechanical dissociation, ventricular tachycardia, or ventricular fibrillation (8).

However, non-HPS hantavirus infection may also occur. In these cases, patients experience nonspecific viral symptoms without cardiopulmonary involvement. Symptoms resemble influenza-like illnesses and may mimic several infectious diseases such as Legionnaires' disease, leptospirosis, Mycoplasma infection, and Q fever (5). Because the early manifestations of HPS are very similar to those of non-HPS hantavirus infection, diagnosis during the initial phase of illness can be challenging. Nevertheless, HPS is associated with high mortality, resulting in death in approximately 40% of infected patients (3).

In Europe and Asia, hantavirus infection primarily causes hemorrhagic fever with renal syndrome (HFRS). Patients commonly present initially with fever, chills, headache, back pain, abdominal pain, and nausea. Some patients may also develop blurred vision, conjunctival injection, rash, and facial flushing (6). As the disease progresses, patients may develop internal hemorrhage, hypotension, and acute kidney injury. The mortality rate of HFRS varies depending on the viral strain and generally ranges from approximately 1% to 15% (6).

Although HFRS is usually self-limiting and many patients recover completely, prognosis largely depends on the infecting hantavirus serotype. Infection with the Puumala virus is typically associated with mild disease, whereas Hantaan and Dobrava viruses are associated with more severe disease (6,7). HFRS should be considered in the differential diagnosis of patients presenting with acute kidney injury, particularly in endemic or high-risk geographic regions. Important differential diagnoses include yellow fever, Ebola virus disease, dengue fever, leptospirosis, malaria, murine typhus, septicemia, disseminated intravascular coagulation (DIC), and thrombotic thrombocytopenic purpura (TTP) (7). During patient evaluation, thrombocytopenia is an important laboratory finding associated with increased vascular permeability and disease severity (4).

In the early stages of acute kidney injury (AKI), patients may present with decreased urine output, proteinuria, and hematuria. As the disease progresses into the oliguric phase, serum creatinine levels increase and glomerular filtration rate declines. Severe oliguria or anuria may subsequently result in electrolyte disturbances, including hyperkalemia and hyperphosphatemia (6).

Enzyme-linked immunosorbent assay (ELISA) is commonly used for the detection of hantavirus-specific IgM antibodies

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and is considered a reliable diagnostic method for identifying acute hantavirus infection. ELISA is used in the diagnosis of both HPS and HFRS (5). The humoral immune response may be useful in monitoring disease progression. IgM antibodies appear early in infection and typically reach peak levels approximately 7–11 days after symptom onset, whereas IgG levels increase during recovery (5). Western blot analysis may also be used to identify viral antigens. In contrast, polymerase chain reaction (PCR) testing is considered less reliable because the period of detectable viremia is typically brief (5).

There is currently no specific antiviral treatment for hantavirus infection. If HPS is suspected, the patient should receive immediate medical attention, preferably in an intensive care unit (ICU), even before the diagnosis is confirmed (2). Early aggressive supportive care is essential because patients with acute HPS may rapidly progress to severe respiratory failure, hemodynamic instability, and death (2,8). Patients with suspected HPS should receive empiric broad-spectrum antibiotic therapy while awaiting definitive diagnosis, particularly to cover alternative infectious etiologies with similar clinical presentations. Supportive management should also include antipyretics and analgesics as needed (8). The cardiopulmonary dysfunction associated with HPS is believed to result primarily from intense inflammatory responses and increased vascular permeability mediated by circulating cytokines (4). Early initiation of extracorporeal membrane oxygenation (ECMO) in patients with severe cardiopulmonary decompensation has been associated with survival rates approaching 80% (8).

Poor prognostic indicators in HPS include plasma lactate levels greater than 4.0 mmol/L and a cardiac index below 2.2 L/min/m². Pulmonary edema and pleural effusions are common manifestations, whereas multiorgan dysfunction syndrome is relatively uncommon. During the convalescent phase, survivors frequently develop polyuria followed by rapid clinical improvement (8).

Management of HFRS depends on the stage of illness and the patient's hemodynamic status. Early and carefully monitored fluid resuscitation remains the cornerstone of supportive management. In patients with shock, intravenous albumin administration and vasoactive agents may be beneficial. However, fluid therapy must be administered cautiously because increased capillary permeability may lead to fluid extravasation and pulmonary edema. Diuretics may be useful in patients with oliguria and volume overload. If conservative management fails, renal replacement therapy should be considered, especially in patients with refractory volume overload, metabolic acidosis, or hyperkalemia (6).

Patients with suspected or confirmed hantavirus infection should be managed using appropriate infection prevention and control precautions. If feasible, placement in a negative-pressure isolation room may be considered during aerosol-generating procedures (3).

Emergency department personnel should receive training on the early recognition of hantavirus infection to facilitate prompt implementation of infection control measures, including appropriate use of personal protective equipment (PPE) (3).

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1.3. Conflict of interest

There is no conflict of interest to declare.

1.4. Using artificial intelligence chatbots

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